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# Benefits of Using Hydroxychloroquine/Chloroquine in the Treatment of Immunomediated Rheumatic Diseases

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**KEYWORDS:**

Hydroxychloroquine, Chloroquine, Immune-mediated rheumatic diseases, Systemic lupus erythematosus, Immunomodulation.

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**ABSTRACT**

Antimalarial drugs, particularly hydroxychloroquine (HCQ) and chloroquine (CQ), have been used for decades as cornerstone therapies in the management of immune-mediated rheumatic diseases (IMRDs). Beyond their classical role in disease activity control, these agents exert pleiotropic effects encompassing immunomodulation, anti-inflammatory signaling, antithrombotic activity, and metabolic and cardiovascular protection. Robust clinical evidence supports their ability to reduce disease flares, limit cumulative organ damage, improve long-term survival, and decrease infection risk, especially in systemic lupus erythematosus (SLE). This review provides an in-depth and updated analysis of the mechanisms of action, clinical applications across autoimmune diseases, metabolic and vascular effects, safety profile, and emerging therapeutic perspectives of HCQ and CQ, highlighting their enduring relevance and expanding role in contemporary rheumatology and systemic medicine.

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## INTRODUCTION

Hydroxychloroquine (HCQ) and chloroquine (CQ) are synthetic 4-aminoquinoline compounds initially developed as antimalarial agents but subsequently repurposed for the treatment of immune-mediated rheumatic diseases (IMRDs), including systemic lupus erythematosus (SLE) [1] and rheumatoid arthritis (RA) [2]. Their incorporation into rheumatologic practice represents one of the earliest and most successful examples of therapeutic repositioning, driven by consistent observations of anti-inflammatory and immunoregulatory effects.

Although HCQ and CQ share similar pharmacokinetic and pharmacodynamic properties, HCQ is currently preferred in clinical practice due to its more favorable safety profile, particularly regarding retinal toxicity [3,4]. Consequently, HCQ is universally recommended as background therapy in SLE and is frequently employed as adjunctive treatment in other IMRDs.

Importantly, antimalarial drugs are not merely symptomatic agents. Long-term observational and cohort studies have demonstrated their association with reduced cumulative organ damage [5,6], improved overall survival [7,8], and decreased rates of thrombosis and severe infections [9]. These benefits suggest disease-modifying properties that extend beyond conventional immunosuppression.

Despite extensive clinical experience, the mechanisms underlying these broad therapeutic effects remain incompletely elucidated. Current evidence indicates a multifactorial mode of action involving modulation of innate and adaptive immune responses, interference with intracellular signaling pathways, and downstream metabolic and vascular effects. Together, these properties position HCQ and CQ as unique immunomodulatory agents with systemic benefits relevant across multiple autoimmune and inflammatory conditions.

## TREATMENT OF AUTOIMMUNE DISEASES

### Systemic Lupus Erythematosus (SLE)

Antimalarial drugs constitute a foundational pillar of SLE management and are recommended for virtually all patients unless contraindicated [1]. HCQ and CQ have consistently demonstrated efficacy in reducing global disease activity, preventing disease flares, and enabling glucocorticoid-sparing strategies [12]. Their benefits extend across multiple organ systems, including cutaneous, musculoskeletal, and hematological manifestations [13,14].

Although antimalarials have not historically been classified as primary therapy for lupus nephritis, accumulating evidence supports a renoprotective effect. Observational studies indicate that HCQ use is associated with delayed progression of renal damage and improved long-term renal outcomes, independent of immunosuppressive regimens [15].

Sustained antimalarial therapy has also been associated with reduced damage accrual across diverse organ domains [5,6], improved survival [7,8], and lower rates of major infections [16]. In addition, HCQ exerts favorable metabolic and vascular effects, including improvements in lipid and glucose metabolism and a reduction in thrombotic events [9]. These pleiotropic actions are particularly relevant given the heightened cardiovascular risk inherent to SLE.

### Rheumatoid Arthritis (RA)

In RA, antimalarial drugs are primarily used as adjunctive therapy, most notably as part of combination regimens with conventional disease-modifying antirheumatic drugs (DMARDs). The classic triple therapy combining methotrexate, sulfasalazine, and HCQ has demonstrated significant efficacy, particularly in early or less aggressive disease [2].

HCQ improves clinical symptoms and laboratory markers of inflammation, especially in patients with early and mild RA [17]. However, unlike other DMARDs, HCQ does not appear to significantly inhibit radiographic progression, underscoring its role as a complementary rather than structural-modifying agent.

Notably, HCQ confers important metabolic and cardiovascular benefits in RA, including reductions in total cholesterol and triglyceride levels, improved glycemic control, and a lower incidence of diabetes mellitus [18,19]. These effects may partially offset the increased cardiovascular morbidity associated with chronic inflammatory arthritis.

### Sjögren Disease (SjD)

In primary Sjögren disease, HCQ and CQ have demonstrated modest but clinically meaningful benefits. Clinical studies suggest improvements in sicca-related oral symptoms, increased unstimulated salivary flow, and reductions in systemic inflammatory markers such as ESR and CRP [20].

Immunologically, antimalarial therapy has been associated with decreased levels of IgM, IgA, and rheumatoid factor. Furthermore, HCQ may reduce the frequency and severity of extraglandular manifestations, including arthritis, fatigue, purpura, Raynaud's phenomenon, and hypergammaglobulinemia [21]. Emerging evidence also suggests a potential cardioprotective role, reinforcing the systemic benefits of therapy in this condition.

### Antiphospholipid Syndrome (APS)

In secondary APS, particularly when associated with SLE, HCQ exerts a well-established protective effect against thrombotic events [9,22]. This benefit appears to be mediated through antiplatelet, endothelial, and immunological mechanisms rather than classical immunosuppression alone. Additional metabolic effects, including improvements in lipid and glucose profiles, further contribute to vascular risk reduction in this high-risk population.

### Sarcoidosis

HCQ and CQ have demonstrated efficacy in selected forms of sarcoidosis, particularly cutaneous, articular, neurological, and pulmonary involvement [23]. Their use is especially valuable in patients with chronic disease or contraindications to prolonged glucocorticoid therapy, offering a steroid-sparing alternative with a favorable safety profile.

## ANTIVIRAL PROPERTIES

Antimalarial drugs have long been recognized for their in vitro antiviral activity, mediated primarily through alkalinization of endosomal compartments and interference with viral entry and replication. During the COVID-19 pandemic, HCQ demonstrated inhibitory effects against SARS-CoV-2 in vitro [24].

However, subsequent clinical studies failed to demonstrate consistent clinical benefit. Current evidence does not support the use of HCQ for the treatment or prophylaxis of COVID-19, and its routine use for viral infections cannot be recommended outside of controlled research settings.

## METABOLIC AND CARDIOVASCULAR EFFECTS

HCQ exerts clinically relevant metabolic effects that extend beyond inflammation control. These include improved insulin sensitivity, reduced peripheral insulin resistance, and modulation of pancreatic insulin secretion [18]. Collectively, these effects translate into a lower incidence of diabetes mellitus among patients receiving long-term HCQ therapy.

HCQ also favorably influences lipid metabolism by reducing hepatic cholesterol synthesis through inhibition of lysosomal function, impairing lysosomal cholesterol transport, and altering intracellular lipid processing [19]. These mechanisms contribute to improved lipid profiles and may partially explain the observed reduction in cardiovascular events among treated patients.

## IMMUNOMODULATORY ACTION

The immunomodulatory effects of antimalarial drugs are central to their therapeutic efficacy in IMRDs. HCQ and CQ inhibit lysosomal activity in phagocytes and macrophages, impairing antigen processing and presentation [10]. They also suppress T-cell activation and reduce the production of key pro-inflammatory cytokines, including IFN-γ, TNF, IL-1, and IL-6.

A particularly important mechanism involves inhibition of Toll-like receptors (TLR) 7 and 9, especially within plasmacytoid dendritic cells, leading to reduced IFN-α production [11]. Additional effects include inhibition of the cGAS–STING pathway, suppression of phospholipase A2 activity, and stimulation of endothelial nitric oxide production, resulting in antiproliferative and vasoprotective effects [25].

## ANTITHROMBOTIC ACTION

The antithrombotic properties of HCQ have been recognized for several decades [26,27]. Early clinical observations, including those by Charnley, highlighted its utility in preventing postoperative thromboembolic events following total hip arthroplasty [28].

Mechanistically, HCQ inhibits platelet aggregation in a dose-dependent manner, reduces arachidonic acid production in activated platelets, and interferes with antiphospholipid antibody–mediated endothelial and platelet activation [9,25]. These complementary mechanisms collectively contribute to its protective effect against arterial and venous thrombosis.

## OTHER THERAPEUTIC BENEFITS

Given their antimalarial origin, HCQ and CQ retain activity against certain infectious agents [16]. Notably, antimalarial therapy in autoimmune disease has been associated with a reduced risk of infections, contrasting with most immunosuppressive treatments that increase susceptibility [29].

Observational studies have also suggested a potential protective effect against malignancies in SLE patients receiving HCQ [29]. Furthermore, HCQ has been incorporated into experimental oncologic regimens, based on its ability to disrupt lysosomal function and autophagy, thereby enhancing the efficacy of conventional chemotherapeutic agents [30].

Table 1 and Figure 1 summarize the main benefits of HCQ in rheumatic diseases.

## TOXICITY

Antimalarial drugs are generally safe and well tolerated. Gastrointestinal adverse effects are the most frequently reported [31], while cutaneous reactions range from mild rashes to severe dermatologic syndromes [32,33].

Retinopathy, although rare, represents the most clinically significant long-term toxicity. When recommended dosing guidelines are followed, the risk remains low within the first decade of use [4]. Cardiac toxicity, neuromyopathy, neuropsychiatric manifestations, and hemolysis in G6PD-deficient patients are uncommon but potentially serious adverse events [34–37].

## PREGNANCY AND LACTATION

HCQ and CQ are safe and recommended during pregnancy and lactation in patients with SLE [38]. Discontinuation is associated with increased disease activity and flare risk during gestation [39].

Importantly, HCQ use is associated with a reduced risk of recurrent neonatal cardiac lupus in anti-Ro/SSA– and anti-La/SSB–positive mothers [40,41], supporting its continued use throughout pregnancy.

## CONCLUSION

Antimalarial drugs, particularly HCQ, remain among the most effective, safe, and cost-efficient therapies in modern rheumatology. Their broad spectrum of immunological, metabolic, antithrombotic, and cardioprotective effects translates into meaningful reductions in organ damage and improved survival, especially in SLE. Emerging evidence suggests that their therapeutic potential extends beyond rheumatology, warranting continued investigation into novel clinical applications across systemic and chronic diseases.

## DECLARATIONS

### Conflict of Interest

The authors declare that they have no conflicts of interest related to the content of this manuscript.

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**Authors' Contributions**

Jozélio Freire de Carvalho conceived the study, performed the literature review, interpreted the data, and drafted the manuscript. Mariana Fernandes Carneiro contributed to the literature review, critical revision of the manuscript, and approved the final version.

All authors read and approved the final manuscript.

**Ethics Approval and Consent to Participate**

Not applicable. This manuscript is a narrative review based on previously published studies and does not involve human participants or experimental animals.

**Consent for Publication**

Not applicable.

**Availability of Data and Materials**

Not applicable.

**Use of Artificial Intelligence**

Artificial intelligence–assisted tools were used during the preparation of this manuscript exclusively to support language refinement, organization of scientific content, and editorial consistency. The use of artificial intelligence did not involve data generation, data analysis, image creation, or interpretation of results. All scientific content, critical analysis, interpretation of the literature, and final editorial decisions were performed by the authors, who take full responsibility for the accuracy, integrity, and originality of the work.

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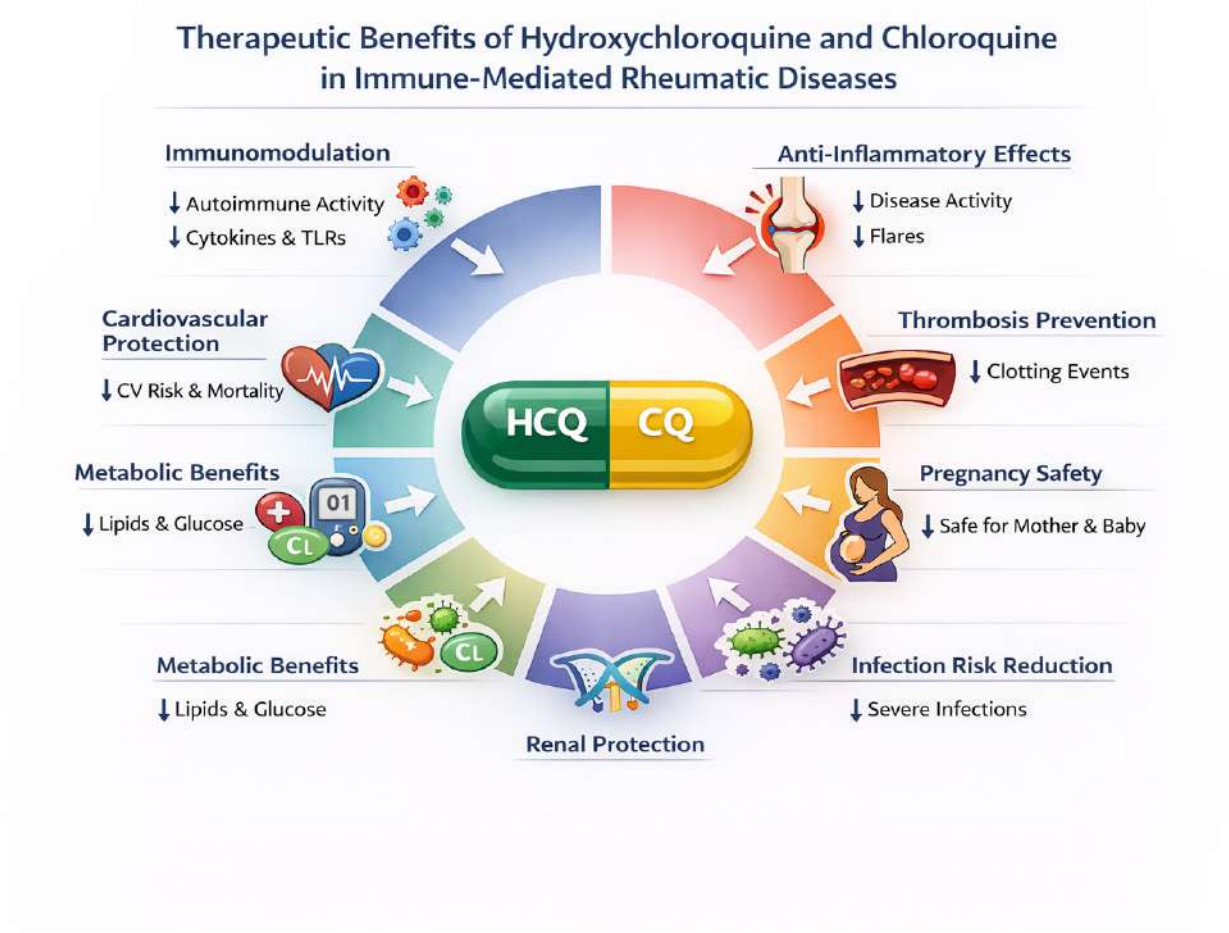
**Table 1. Summary of the main therapeutic effects of hydroxychloroquine and chloroquine in immune-mediated rheumatic diseases.**

| Domain | Key effects of HCQ/CQ | Clinical relevance | Main diseases involved |
|---|---|---|---|
| **Immunomodulatory action** | Inhibition of lysosomal function; reduced antigen presentation; suppression of T-cell activation; inhibition of TLR7/9 signaling; reduced IFN-α, TNF, IL-1, IL-6; modulation of cGAS–STING pathway | Reduction of immune activation and chronic inflammation; disease-modifying effect | SLE, RA, Sjögren disease, APS |
| **Anti-inflammatory effects** | Decreased production of pro-inflammatory cytokines; attenuation of innate and adaptive immune responses | Lower disease activity; prevention of flares; glucocorticoid-sparing effect | SLE, RA, Sjögren disease |
| **Antithrombotic action** | Dose-dependent inhibition of platelet aggregation; reduced arachidonic acid production; interference with antiphospholipid antibody–mediated activation | Reduction of arterial and venous thrombotic events | SLE, secondary APS |
| **Metabolic effects** | Improved insulin sensitivity; reduced peripheral insulin resistance; lower incidence of diabetes mellitus | Improvement of metabolic profile; reduction of cardiovascular risk | SLE, RA |
| **Lipid profile modulation** | Reduced hepatic cholesterol synthesis; altered lysosomal cholesterol transport | Lower total cholesterol and triglyceride levels | SLE, RA |
| **Cardiovascular protection** | Combined anti-inflammatory, antithrombotic, and metabolic effects | Reduction of cardiovascular morbidity and mortality | SLE, RA, Sjögren disease |
| **Renal protection** | Delayed progression of renal damage; reduced damage accrual | Improved long-term renal outcomes | Lupus nephritis |
| **Infection risk modulation** | Lower incidence of major infections compared with other immunosuppressive therapies | Improved long-term safety profile | SLE, IMRDs |
| **Cutaneous and extraglandular manifestations** | Improvement of skin lesions, arthritis, fatigue, purpura, Raynaud's phenomenon | Better symptom control and quality of life | SLE, Sjögren disease, Sarcoidosis |
| **Safety profile** | Generally well tolerated; rare serious toxicity with recommended dosing and monitoring | Long-term treatment feasibility | All IMRDs |
| **Pregnancy and lactation** | Safe use; reduced risk of disease flares and neonatal cardiac lupus | Improved maternal and fetal outcomes | SLE, APS |
| **Emerging and adjunctive uses** | Antiviral activity in vitro; autophagy inhibition; oncologic adjuvant potential | Experimental and investigational applications | Infectious diseases, oncology |

**Abbreviations:** APS, antiphospholipid syndrome; CQ, chloroquine; cGAS–STING, cyclic GMP–AMP synthase–stimulator of interferon genes; HCQ, hydroxychloroquine; IFN, interferon; IL, interleukin; IMRDs, immune-mediated rheumatic diseases; RA, rheumatoid arthritis; SLE, systemic lupus erythematosus; TLR, Toll-like receptor; TNF, tumor necrosis factor.


Therapeutic Benefits of Hydroxychloroquine and Chloroquine in Immune-Mediated Rheumatic Diseases
Immunomodulation
↓ Autoimmune Activity
↓ Cytokines & TLRs
Anti-Inflammatory Effects
↓ Disease Activity
↓ Flares
Cardiovascular Protection
↓ CV Risk & Mortality
Thrombosis Prevention
↓ Clotting Events
HCQ
CQ
Metabolic Benefits
↓ Lipids & Glucose
Pregnancy Safety
↓ Safe for Mother & Baby
Metabolic Benefits
↓ Lipids & Glucose
Infection Risk Reduction
↓ Severe Infections
Renal Protection